Conclusions

The most important aspect of the treatment of concealed accidental haemorrhage is the treatment of shock.

Transfusion of incompatible blood and intravenous injection of uncontrolled quantities of blood and other fluids contribute to the mortality of these patients.

Routine administration of continuous oxygen, avoidance of surgical treatment capable of causing shock, and care with intravenous therapy will reduce the incidence of anuria.

Certain complications, notably post-partum haemorrhage and anuria, should be anticipated, and be promptly treated if they occur.

There is no place in treatment for artificial rupture of the membranes in the absence of contractions, plugging of the vagina, version, dilatation of the cervix, application of vulsellum to the scalp, and other radical procedures.

Artificial rupture of the membranes, even in the presence of contractions, is dangerous without preliminary treatment of shock.

Transverse lower-segment caesarean section has a small but definite place in treatment, the indication being the continued absence of contractions and progressive deterioration of the patient's condition after conservative measures have been given a fair trial.

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POST-DYSENTERIC COLITIS*

BY

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For many years it has been known that a primary attack of dysentery may be followed by a chronic disorder of the colon. In amoebiasis this disorder is the rule rather than the exception, and it may take the form of periodic dysenteric attacks or simply of chronic diarrhoea. In a much smaller proportion of cases, acute bacillary infections may be followed by chronic dysentery. The diagnosis of one or other of these well-known conditions, however, always depends upon the recovery of the causal organism from the stools or from lesions in the colon, and it is usually inferred that the principal factor in the "colitis" is the persistence of that organism.

The present paper deals with a number of dysenteric and post-dysenteric cases seen by me at the close of the 1939–45 war. In a proportion of these cases disorder of the colon persisted long after the removal of the dysenteric organism. This disorder, which may be termed post-dysenteric colitis (Hurst, 1943), seemed to be of two main types—ulcerative and non-ulcerative. The two types could be clearly differentiated for prognosis, treatment, and eligibility for pension.

The background to this report can be found in the numerous independent studies made during and after the 1939–45 war on the prevalence of dysentery in tropical campaigns, and in particular in papers by Adams (1945), Hargreaves (1946), and Stewart (1947a, 1947b, 1948). The present survey comprised 228 cases of dysentery and diarrhoea admitted to a hospital in Ceylon (Combined Services Hospital, Trincomalee) and 246 cases observed in the Tropical Diseases Centre, Smithdown Road Hospital, Liverpool. Details of the bacteriological and other technical methods employed in the investigation have already been described (Stewart, 1947a, 1948).

Cases Seen in Ceylon

The principal known causes of acute diarrhoea and dysentery in the Trincomalee area of Ceylon are shown in Table I. As might be expected, the majority of the cases

TABLE I.—*Dysenteric Disorders in Ceylon* (European and Asiatic Hospital Admissions)*

	Actual Numbers	% of Total
Amoebiasis	314	50
Bacillary dysentery	230	37
Flagellate dysentery	69	11
Salmonella infections	14	2

* Excluding helminthic infections, typhoid fever, undiagnosed or symptomatic diarrhoea.

were accounted for by intestinal amoebiasis (50%) and bacillary dysentery (37%). For various reasons it was

*This investigation formed part of a research programme carried out at the Liverpool School of Tropical Medicine for the Colonial Medical Research Committee.

impossible to follow up many of these cases, and the analysis of relapses and recurrences of diarrhoea among cases of intestinal amoebiasis and bacillary dysentery was made from a smaller group (Table II).

TABLE II.—*Relapses and Recurrences of Diarrhoea in a Group of Cases of Amoebiasis and Bacillary Dysentery (Ceylon, 1945–6)*

Group	No. Treated	Recurrences After Treatment			
		Within 3 Weeks		Within 3 Months*	
		Organism	Diarrhoea (With or Without Organism)	Organism	Diarrhoea (With or Without Organism)
Bacillary dysentery (123)					
Sh. shigae	12	1 (1 death)	1	—	
Sh. shmitzi	6	—	—	—	Four return cases, each with *E. histolytica*
Sh. flexneri	30	—	—	—	
Sh. sonnei	26	16	6	6	
Others	10	—	—	—	
Diagnosed by exudate	39	—	1	—	
Intestinal amoebiasis (100)					
Dysentery	54	2	10	4	17
Chronic diarrhoea	46	—	8	2	12
Dual infection	5	—	—	2	2

* Including cases which relapsed after further courses of treatment.

In bacillary dysentery adequately treated with sulphonamides the relapse rate was negligible. *Shigella sonnei* displayed its well-known tendency to linger in the bowel beyond the period of treatment, but produced no further disease. Even in the absence of treatment, acute bacillary dysentery was noted to be self-limiting in a few cases admitted to hospital on the third or fourth day of illness. The findings therefore, in agreement with those of other investigators (Fairbrother, 1944; Scadding, 1945), suggest that chronic bacillary dysentery of the type described by Manson-Bahr (1943) and Rogers (1944) after the 1914–18 war must be extremely uncommon nowadays.

In contrast, there were many recurrences of symptoms among patients treated for active intestinal amoebiasis.* Not all of these recurrences were accounted for by persistence of *Entamoeba histolytica,* though it is obvious that such a statement is valid only when repeated attempts have been made to detect the parasite. In the present investigation it was assumed that stool examinations on 12 successive days, together with sigmoidoscopic scrapings and flotation technique, sufficed to identify the parasite in about 90% of cases (Faust *et al.*, 1939; Kershaw, 1946; Kershaw, O'Meara, and Stewart, 1948). With these criteria fulfilled it is evident from Table II that diarrhoea persisted in the absence of *E. histolytica* in 23 out of 29 relapses (79%). These cases probably represent the main source of post-dysenteric colitis.

*The standard treatment in this series was the Liverpool method (Adams, 1945). Each patient was treated for three weeks or more with emetine bismuth iodide, chiniofon ("quinoxyl"), and acetarsol ("stovarsol"). Injections of 1 gr. (65 mg.) of emetine hydrochloride (up to 12) were given to dysenteric or hepatic cases before the three-weeks course.

Cases Seen at the Tropical Diseases Centre, Smithdown Road Hospital, Liverpool

In the Liverpool area a number of cases of acute or chronic diarrhoea occurring in Service or Merchant Navy personnel returning from the Tropics were referred to the Tropical Diseases Centre, attached to the Liverpool School of Tropical Medicine. The admissions for 18 months in 1947–8 supported the evidence contained in the Ceylon data, in that the majority of the cases were accounted for by amoebiasis (Table III). The incidence of bacillary dysentery was negligible, and cases of chronic bacillary infection were again absent. The Liverpool hospital admissions, however, contained a group not definitely represented among the earlier cases seen in Ceylon: 49 cases, which we have labelled post-dysenteric colitis, in which diarrhoea persisted in the absence of any recognized intestinal pathogen.

TABLE III.—*Cases of Acute and Chronic Diarrhoea in Service Patients or Merchant Seamen Returning to England from the Tropics (Hospital Admissions to the Tropical Diseases Centre, Smithdown Road Hospital, Liverpool)*

	Actual Numbers	% of Total
Bacillary dysentery (acute, 2)	2	0·8
Intestinal amoebiasis (dysentery, 66; chronic diarrhoea, 108)	174	70·7
Other infections	21	8·5
Post-dysenteric colitis	49	20·0
Total	246	

TABLE IV.—*The Main Clinical Features of 49 Cases of Post-dysenteric Colitis*

History			
Bacillary dysentery	12	Previous diarrhoea	49
Amoebiasis	28	Intermittent diarrhoea	19
Both	9	Constipation	—
Symptoms			
Abdominal pain	26	Continuous diarrhoea	30
Dyspepsia	13		
Lassitude	25		
Loss of weight	23		
Good health	15		
Sigmoidoscopic Findings		**Faeces**	
Normal	18	Loose. No exudate	18
Granular areas	9	Loose. No exudate or scanty mucoid exudate	9
Ulcers	18	Blood and pus present	22
"Plush"	4		

The symptoms associated with post-dysenteric colitis are shown in Table IV. The condition defines itself in a patient with a proved history of dysentery, persistence of diarrhoea, and absence of the original infecting organism. As in most other forms of colitis, the main complaint was diarrhoea, which varied in severity and in frequency: in 62% it was continuous and in 38% intermittent. In 30% of the cases there was no complaint other than diarrhoea. The remainder gave histories or showed signs of lassitude, loss of weight, and abdominal pain, sometimes dyspeptic in character but more often "colicky."

Sigmoidoscopic and stool examinations showed that the cases could be divided into two main types (Table V). In

TABLE V.—*Provisional Classification of 49 Cases of Post-dysenteric Colitis*

No. of Cases	Condition of Colon	Region of Colon Most Affected	Other Findings	Type of Case (Provisional Classification)	
18	Normal or spastic. Mucosa normal. No exudate	—	General condition usually good. Neurotic tendency present	Neurosis	Type I: Functional post-dysenteric colitis
9	Wall normal or spastic. Granular areas on mucosa, formed of small pitted scars, pin-point depressions, and patchy hyperaemia. Mucoid exudate	Rectosigmoid junction	General condition good	"Irritable colon"	
18	Areas of ulceration. Intervening mucosa oedematous and congested. Severe diarrhoea with blood and pus in stools	Rectosigmoid junction	General condition variable. Occasionally leucocytosis and serum agglutinins for coliform organisms	(*a*) Added bacterial infection	Type II: Ulcerative post-dysenteric colitis
4	Entire mucosa intensely congested and oedematous. Superficial sloughing. Copious exudate of blood and mucopus	Entire area visualized by sigmoidoscopy	General condition poor. Anaemia	(*b*) Aetiology unknown	

one type (55%) the mucosa of the colon was normal in appearance, and the stools, though loose, contained no exudate or a scanty mucoid exudate. In these cases the diarrhoea was usually intermittent and seldom severe ; in some cases a few loose or precipitate stools were passed in the morning only. In other cases neurotic or anxiety traits were noted, and these, together with the absence of organic change in the colon, suggested that the condition was to a large extent functional (see Case A). In most cases there was a slow natural improvement, without treatment. Four cases, rather more refractory, seemed to benefit from treatment with antispasmodics (hyoscine hydrobromide, 1/200 gr. (0.32 mg.) six-hourly) and phenobarbitone.

The second but more important type of case (45%) showed inflammatory or ulcerative changes in the colon, with blood and pus in the faeces (Table V). These cases were seriously and sometimes intractably ill. It is possible that they required further subdivision into two types, represented by Cases B and C, and defined by the aetiological and therapeutic factors detailed below.

Documentary records showed that each of these 49 cases had a previous history of dysentery and had experienced repeated attacks of diarrhoea before admission to hospital. The majority (57%) had suffered from amoebic dysentery, 24.5% from bacillary dysentery, and 18.5% from a dual infection. Acton (1933) and Silverman and Leslie (1945) have attributed the patency and severity of amoebic dysentery in tropical areas to concomitant infection with Shigella organisms. On the other hand, Stewart, O'Meara, and Kershaw (1948) have shown that the occurrence of the dysenteric state in amoebiasis is not necessarily dependent upon a coincident bacillary infection, and that mixed infections are not necessarily more severe or more prone to relapse than "unmixed" amoebic infections.

It is worth recalling that the exudate in a bacillary infection is alkaline, whereas that in amoebic dysentery is acid (Stewart, 1948), and it may be that the tissue reaction provoked by the activity of one organism is unfavourable for the simultaneous activity of the other. These findings, together with the absence of chronicity among the Shigella infections in the present series, suggest that the establishment of the post-dysenteric state does not depend upon a previous mixed infection or upon the persistence of a Shigella organism. A proportion (24.5%) of our cases, however, had histories of one or more attacks of bacillary dysentery, with no past or present evidence of amoebiasis, and it is possible that in such cases some additional factor may lead to the development of a colitis after the elimination of the specific Shigella organism.

The additional factor is one which may be present in cases of amoebiasis as well as in bacillary dysentery. In previous communications (Stewart, 1947b, 1948) I have shown that in severe and relapsing cases of amoebiasis there are changes in the relative distribution of the various organisms in the bacterial flora of the colon, and that there is a significant increase in the presence of paracolon organisms ; this group of organisms has often been associated with outbreaks of diarrhoea and "non-specific enteritis," and it is possible that the persistence of diarrhoea in some post-dysenteric cases may be related to the presence of such potential pathogens in the intestine (Table VI).

At this stage the difference beween the two types of cases must be re-emphasized (see Table V). One type developed a mild intermittent diarrhoea with little or no inflammatory change in the colon and, at most, a scanty mucoid exudate in the faeces, while the second type developed an ulcerative colitis with severe and persistent diarrhoea and a purulent exudate. It is in the second or

TABLE VI.—*Ulcerative Post-dysenteric Colitis*

Case No.	History	Bacterial Flora of Faeces*	Serum Agglutination Titre	Treatment	Result
1	Bacillary dysentery	Enterococci	Not done	Penicillin (enemata) 10 × 10^6 units	Improved‡
2	Amoebiasis	*Bact. coli* Enterococci	Negative Not done	Chiniofon. Sulphasuxidine Penicillin 8 × 10^6 units i.m. and sulphathiazole 52 g.	No change
3	,,	*Bact. coli* Enterococci Paracolon	Negative Not done Negative	Penicillin (enemata) 10^6 units × 10	Improved
4	,,	*Bact. coli* Enterococci *Bact. morgani*	(O) 100 Not done Negative	Penicillin 8 × 10^6 i.m. Sulphasuxidine 60 g.	Slow improvement
5	,,	*Bact. coli* Enterococci Paracolon†	,, ,, (O) 100	Penicillin (enemata) 10 × 10^6	Improved
6	,,	*Bact. coli* Enterococci Paracolon†	Negative Not done (O) 50 (H) 200	Penicillin (enemata) Penicillin 8 × 10^6 i.m. and sulphathiazole 52 g.	No change Rapid improvement
7	,,	*Bact. coli* Paracolon	Negative ,,	Chiniofon—2½% retention enemata	Slow improvement
8	Bacillary dysentery Amoebiasis	*Bact. aerogenes* Paracolon	,, (O) 50	Chiniofon 2½% R.E. Penicillin R.E. 10 × 10^6	No change Improvement
9	Bacillary dysentery Amoebiasis	*Bact. coli* *Bact. aerogenes* Enterococci	Not done ,, ,, ,, ,,	Penicillin 8 × 10^6 i.m. and sulphathiazole 52 g.	Improvement
10	Bacillary dysentery Amoebiasis	*Bact. coli* Paracolon	Negative ,,	Penicillin R.E. 10 × 10^6	Improvement
11	Amoebiasis	*Bact. coli* Enterococci	(O) 50 (H) 200 Not done	Penicillin 8 × 10^6 i.m. and sulphathiazole 52 g. Sulphasuxidine 60 g.	Improvement
12	,,	*Bact. coli* *Bact. aerogenes* Paracolon Enterococci	(O) 50 Negative ,, Not done	Penicillin 8 × 10^6 i.m. and sulphathiazole 52 g. "Benadryl"	No change ,, ,,
13	,,	*Bact. coli* Paracolon	(H) 50 (H) 200	Chiniofon 2½% R.E.	Slow improvement

* Excluding Clostridia. † Eliminated after treatment. ‡ Improved = lessening of diarrhoea, diminution of exudate, healing of lesions (sigmoidoscopy).

"ulcerative" type (Table VI) that the element of added bacterial infection, non-specific in type and bearing some relation to the factor already described in cases of relapsing amoebiasis, might play a part. The number of cases in the present series, however, is too small for a statistical estimate of the significance of this factor, and it must also be recognized that changes in the intestinal flora *per se* do not necessarily constitute a pathogenic influence upon the colon (Stewart, 1948 ; Stewart, Jones, and Rogers, 1948).

In some cases it was possible to demonstrate serum agglutinins against coliform and paracolon organisms isolated from the faeces (Table VI). Blood cultures, taken before and after sigmoidoscopy, were negative in ten cases. Leucocyte counts varied from normal to 24,000 cells per c.mm. In other words, there was no definite evidence of a characteristic systemic response to the infection, just as there was no specific pathogen which could be incriminated. It would seem, nevertheless (Table VI), that some improvement might be expected in such cases from the use of penicillin and sulphonamides ; the bacteria concerned, including coliform Gram-negative organisms, can be inhibited by high concentrations of these agents *in vitro* (Stewart, 1947c) and in experimental amoebiasis *in vivo* (Stewart and Jones, 1948), while penicillin has been shown to retain bacteriostatic potency in the colon (O'Connor, 1947). If penicillin and sulphonamides fail, some improvement may follow the use of a 2½% retention enema of chiniofon, which has antibacterial properties and produces a decrease in the number of coliform organisms in the faeces.

In the series described there were four cases showing pronounced inflammatory changes in the colon in which antibacterial treatment was of no avail, notwithstanding the use of large doses of soluble and insoluble sulphonamides, penicillin, and chiniofon. These cases were virtually intractable. One case showed unexplained remissions, but the others became gradually worse. The mucosa of the colon was evenly oedematous, congested, and fragile, like velvet plush in appearance. Large quantities of fresh blood were passed rectally. The patients grew weak, lost weight, and developed a hypochromic anaemia. No skin lesions, glossitis, or hypoproteinaemia were observed. Leucocyte counts were within normal limits. Skin tests and serum agglutination reactions with intestinal coliforms were negative, as were blood cultures. No definite benefit was seen after treatment with supplementary vitamins, high-protein diet, haematinics, and blood transfusion. On the hypothesis that the condition might be maintained by a sensitization mechanism, two cases were treated with antihistamine drugs, but without improvement.

It is obvious that such cases resembled the condition of idiopathic ulcerative colitis in their clinical course and intractability ; the only difference was that they arose as direct sequelae to amoebic dysentery, the transition from one condition to the other being well exemplified by Case C.

In the cases here described the symptoms were pronounced enough to necessitate admission to hospital for investigation if not for treatment. It is probable that milder forms of post-dysenteric colitis are very common indeed among personnel returning from active service in certain areas of the Tropics. The majority of these cases may be expected to show the slow natural improvement of type I, but it is important that the more serious inflammatory conditions of type II should be recognized at the earliest possible stage.

Illustrative Cases

Case A : Post-dysenteric Colitis, Type I.—Bacillary and amoebic dysentery in India in 1941. Recurrent diarrhoea since then, treated in Army hospitals with courses of emetine and sulphaguanidine. On admission slightly underweight, but general condition good. Anxious disposition ; 5–10 bowel movements a day ; stools unformed, but no blood or mucus—12 specimens negative for *E. histolytica,* and 3 negative for bacterial pathogens. Sigmoidoscopy showed mucosa congested ; granular areas at rectosigmoid junction ; scraping gave epithelial cells only. *Treatment :*—Hyoscine hydrobromide (1/200 gr. (0.32 mg.) six-hourly) and phenobarbitone (½ gr. (32 mg.) t.d.s.) for six days. No change. Without treatment, patient's condition began to improve three months later : 3 bowel movements a day ; had gained weight and felt better.

Case B : Post-dysenteric Colitis, Type II (a).—Amoebic dysentery in Egypt in 1942, treated with emetine and chiniofon. Three subsequent relapses similarly treated. Intermittent diarrhoea 1945–6. In 1947 diarrhoea became severe and continuous. On admission he was pale and fatigued. Temperature 99–100° F. (37.2–37.8° C.). 5–6 bowel movements a day. Stools consisted largely of blood and pus ; 12 stools negative for *E. histolytica.* Culture gave a predominance of Group II paracolon bacteria. Serum agglutinated this organism to 1 : 50 ("O") and 1 : 200 ("H"). Leucocyte count 16,540 (75% polynuclears). Sigmoidoscopy revealed congested mucosa in rectum and lower sigmoid colon, with extensive irregular ulcers, most pronounced at rectosigmoid junction. Scraping : negative for *E. histolytica.* Biopsy of edge of ulcer: mucosa largely replaced by polynuclear cells and lymphocytes. *Treatment :*—Rest in bed ; bland non-residue diet (high protein, high calorie) ; penicillin retention enemata daily for 10 days (1,000,000 units in 100–400 ml. saline). No improvement. Six-day course of penicillin (500,000 units four-hourly for four days and six-hourly for two days, intramuscularly) and sulphathiazole (76 g.). Diarrhoea stopped and exudate diminished one week later. Sigmoidoscopy showed congested mucosa with superficial healing of ulcers. Paracolon bacteria disappeared from stools following treatment. After one month's leave the patient returned symptom-free. Sigmoidoscopy now showed complete healing of lesions. No relapse during succeeding year.

Case C : Post-dysenteric Colitis, Type II (b).—Amoebic dysentery in India in 1943. Several relapses, all treated with emetine, chiniofon, and sulphonamides. Admitted to hospital with amoebic dysentery relapse in 1947. Treated with emetine (5 gr.—0.32 g.), "auremetine" (30 gr.—1.95 g.), acetarsol (120 gr.—7.8 mg.); and chiniofon retention enemata (10 days). Diarrhoea persisted after treatment, and a week later *E. histolytica* trophozoites reappeared in stools. Sigmoidoscopy revealed oedematous friable mucosa from anal margin upwards ; extensive irregular ulcers. *Treatment :*— Sulphasuxidine (60 g.) and chiniofon retention enemata (10 days). *E. histolytica* disappeared, but stools became frankly purulent. Further treatment with sulphathiazole, penicillin, supplementary vitamins, etc., brought no improvement, but a spontaneous remission occurred after three months. This was maintained for six months, and then a mild relapse occurred. Faeces were consistently negative for *E. histolytica* and bacterial pathogens. A leucocyte count was within normal limits. There were no serum agglutinins for intestinal coliforms.

Summary

Amoebic dysentery or, less commonly, bacillary dysentery may be followed by chronic colitis, supervening after the clearance of the original infecting organism.

Thus defined, the condition of post-dysenteric colitis occurs in three forms, differing in prognosis and management, and possibly in aetiology:

Type I.—Non-ulcerative ; large functional element ; slow, natural improvement.

Type IIa.—Ulcerative ; element of non-specific added bacterial infection ; therapeutic response to antibacterial treatment.

Type IIb.—Ulcerative ; no response to antibacterial treatment ; possibly akin to idiopathic ulcerative colitis.

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PROGNOSIS IN BRONCHIAL ASTHMA

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Asthma was recognized before the time of the earliest medical literature, although it was only during the sixteenth century that bronchial asthma began to emerge as a distinct entity. Until this time other types of dyspnoea were confused with bronchial asthma, and indeed it took several centuries for the difference to be generally realized. Asthma was mentioned in the Ebers Papyrus, written 1,500 years B.C. (and so must have been known centuries before this), and in the writings of Homer and Herodotus. Hippocrates and Galen mention it, and Aretaeus of Cappadocia, a contemporary of Galen, gave the first fairly accurate description of an attack of asthma.

Until recent times few of the writers on asthma have made much mention of the prognosis. Henry Hyde Salter devoted the whole of Chapter 15 in his book on asthma, published in 1868, to the prognosis. This, he said, depends on the duration of attacks (rather than their severity), their frequency, the completeness of recovery between them, their apparent tendency (that is, whether they appear to be getting more frequent and more severe, or less frequent and lighter), the length of time asthma has existed, the patient's age, and the presence or absence of organic mischief in heart and lungs. He thought that the prognosis in a patient under 15 years of age is almost invariably good, that there is no disease in which age is more important in the prognosis, and that the chances of recovery are in inverse proportion to the length of time the disease has existed.

After reading about asthma in the literature of all periods, what perhaps impresses one most is how some of the earlier physicians, by painstaking and detailed observation of patients, arrived at conclusions which we are to-day verifying and, indeed, in many cases rediscovering.

Present Investigation

An attempt has been made to analyse and assess the factors which govern the prognosis in bronchial asthma. For this purpose patients who had been under observation for bronchial asthma for ten years or more were chosen—patients who made their first visit to the Cardiff Municipal Asthma Clinic in 1935 and 1936. This clinic is run in conjunction with the municipal hospital, to which doctors can refer any case of bronchial asthma or hay-fever living within the city boundaries. Approximately 200 to 400 new cases are seen each year. A team of doctors investigate the patients, treatment is instituted, and the patients are carefully followed up. The first 80 patients to attend in 1935 and 1936 who could be traced and persuaded to attend for interview were seen personally between April, 1946, and April, 1947, when a detailed history was taken. Of the patients not included in the series the majority could not be traced, and most of the remainder had moved to distances precluding personal interview. In the case of patients who died, the relatives and the doctor attending the last illness were contacted. The cases were thus not selected in any way, and only those which appeared to be definite bronchial asthma were included in the series. A large number of patients whose asthma began in early life brought their parents with them to the interview, and the others had usually filled in any gaps in their own knowledge from their parents before coming.

To be accepted as a case of bronchial asthma a patient had to give a history of dyspnoea which occurred in or was much worse in paroxysms and was associated with a tight feeling in the chest. Patients who had had chronic bronchitis as babies and young children, and in whom paroxysmal dyspnoea developed later, were accepted, as were those in whom, following a typical earlier history, dyspnoea had become chronic, with merely fluctuations in its intensity. The asthma clinic records and any in-patient notes of the cases were consulted. The data obtained in this way were then analysed and an attempt made to assess the factors governing the prognosis in bronchial asthma.

In assessing the end-results the patients were placed in one of four categories—namely, cured, improved, unimproved, and dead. The total number of cases investigated was 80, and the results were : cured, 17 (21%) cases ; improved, 43 (54%); unimproved, 12 (15%); dead, 8 (10%). It is of course difficult to speak of cure in asthma, as the patient may possibly develop symptoms again at some later period. The longer he is free, however, the less likely this is to occur, and the more justified we are in using the term "cure." The criterion used in this investigation for placing the patient in the "cured" group is that no asthmatic symptoms whatever have been experienced for at least four to five years. Despite the stringent criteria for cure in the present series 21% have been "cured" for four to eleven years, and over 70% of this group for periods exceeding seven years (see Table I).

TABLE I.—*Period of Freedom From Asthma in 17 Cured Cases*

4–5 years	1 case	8–9 years	3 cases
5–6 ,,	1 ,,	9–10 ,,	3 ,,
6–7 ,,	3 cases	10–11 ,,	2 ,,
7–8 ,,	4 ,,		

Cases in which a mild attack was experienced at intervals of years or in which occasional tightness of the chest was the only remaining manifestation—that is, cases in which "cure" seemed to have been almost but not quite achieved—were placed in the "improved" group, as were all cases showing any improvement occurring before and maintained up to the time of personal interview. Cases in which there was no improvement and those in which the condition was definitely worse were included in the unimproved group. The last group, of course, contains